Problems in Ventilation in the Immediate Postoperative Period

N. P. GUADAGNI, *M.D.*, San Francisco

• **In the present day practice of surgical anesthesia, drugs and techniques are used which require or lead to cessation of voluntary respiration by the patient. Respiration is then controlled by the anesthesiologist. At the termination of operation many patients do not breathe adequately for variable periods of time. The causes include obstruction, excessive sedation, muscle relaxants, the effect of controlled respiration itself and various miscellaneous factors. A diagnosis is made by taking into consideration the drugs and techniques which have been used and the character of the patient's respiratory efforts, if any. The cause may then be treated. In some cases antidotes are available. However, until truly adequate spontaneous respiration is observed for some time the patient must have his efforts assisted. High oxygen concentrations must not be substituted for adequate ventilation.**

BALANCED ANESTHESIA achieved by the administration of intravenous barbiturates, opiates, muscle-relaxing drugs and nitrous oxide by inhalation, is the most commonly used method in California. The reasons for the prevalence of this method include pleasant induction, minimal postoperative nausea and vomiting, elimination of explosive agents and many patients' fear or dislike of regional anesthesia in general and of spinal anesthesia in particular. In order to produce satisfactory working conditions for surgical procedures in which such a combination of anesthetics is used, the dosages employed frequently lead to respiratory depression of either central or myoneural origin. The agents used intravenously are not excreted by the lungs but continue their depressant activity until eliminated by the slower processes of excretion or destruction. Prolonged respiratory depression or paralysis, which rarely occurred before balanced anesthesia came into wide use, are now the major causes of postoperative respiratory inadequacy.

The causes of inadequate ventilation in the postoperative period can be grouped into five categories: (1) Obstruction to the airway; (2) drug depression of the respiratory center; (3) prolonged effect of muscle-relaxing drugs; (4) "habit apnea," occurring after long periods of controlled respiration; (5) miscellaneous causes such as pulmonary atelectasis, pneumothorax, tight dressings and pain on respiration. Several of these factors may co-exist in the same patient.

Obstruction of the airway anywhere between the nostrils or mouth and the pulmonary alveoli has been the most common cause of hypoxia since the advent of general anesthesia. Maintenance of a patent airway is the first principle of anesthesia taught to all medical students. Nevertheless, patients are still placed in jeopardy by such respiratory obstruction as laryngeal edema, tracheal compression occurring after operations on the neck and even the commonplace blocking of the pharynx by the tongue.

Depression of the respiratory center by drugs is often seen in the postoperative period after balanced anesthesia has been used. The slow rate of elimination of the intravenously administered drugs has been mentioned as a cause. Moreover, the degree of depression of the respiratory center during and after an operation, depends on the balance struck between the depressant effect of drugs on the one hand and the stimulation of pain on the other. Patients who have the severe pain of coronary thrombosis or ureteral colic tolerate large doses of opiates and still continue to breathe adequately. Cessation of the pain may bring on a pronounced slowing of the respiratory rate. A comparable phenomenon may be seen at the end of a painful surgical procedure: A patient who was breathing well during the operation drifts into slower and shallower breathing on termination of the stimulus of pain. This is particularly evident when the main anesthetic agent is a barbiturate. The analgesic properties of barbiturates are so poor that large amounts are necessary to obtain even moderate relief of pain. If repeated doses are used, a reservoir of the drug accumulates in the fatty tissues, with the result that during the postoperative period the respiratory depression is even greater than it was during the operation.[1]

The muscle relaxants are probably the most frequent cause of apnea or inadequate respiration occurring after the last skin suture has been tied. Surgeons have become so accustomed to complete

From the Division of Anesthesiology, Department of Surgery, University of California, San Francisco.

Presented before the Section on Anesthesiology at the 87th Annual Session of the California Medical Association, Los Angeles, April 27 to 30, 1958.

muscular relaxation of the patient while they operate that paralysis of all voluntary muscles, including the intercostals and diaphragm, is common during abdominal operations. Moreover, the greatest degree of relaxation is requested at the time of the wound closure. Partial or complete paralysis of the muscles of respiration in the postoperative period may be due to long-acting relaxants used near the end of the operation, to the total cumulative effect of excessive dosage or, in instances, to unexpectedly prolonged drug action. Much investigation has been done and many theories proposed to explain this last, fortunately small, group.[3,5]

"Habit apnea" is the term used to describe apnea occurring after a period of controlled respiration. The incidence of this respiratory difficulty has increased with the increased use of balanced anesthesia. It may last 15 minutes or more. Depression of the respiratory center by inhibiting stimuli from repeatedly distended pulmonary alveoli (Hering-Breur reflex) is the main cause of such apnea, although narcotic depression or the low carbon dioxide tension of hyperventilation may also be a contributing factor.[2]

For the purpose of discussing diagnosis and treatment, all cases of inadequate ventilation are divided first into those in which apnea occurs and those in which it does not. The latter category includes all patients whose spontaneous respiration is not capable of satisfying the need for oxygen and eliminating carbon dioxide. Clinically, absence of cyanosis is evidence of sufficient arterial oxygen if the patient is not anemic. Usually it is safe to assume that if the patient is not cyanotic while breathing room air, no serious accumulation of carbon dioxide will occur. If the patient's respiratory efforts do not fulfill these requirements, one must determine why and in what manner they are deficient. Examination of the chest and careful observation of the respiratory movements will provide the answer. Signs of obstruction of the air passages, such as noisy respiration and retraction of the soft tissues of the chest wall during inspiration, must be looked for and the obstruction relieved if present. Movements and breath sounds of the two sides of the chest are compared and the position of the trachea in the lower neck is noted to rule out the possibility of pulmonary atelectasis or pneumothorax. The former is treated by thorough suctioning of trachea and bronchi to aspirate mucus and stimulate coughing. The suctioning of plugs of mucus may require direct vision through a bronchoscope. That complete reexpansion of the lung has taken place should be confirmed by a roentgenogram of the chest at the earliest opportunity. Moderate pneumothorax without serious respiratory embarrassment requires no active treatment, although the condition should be kept under observation until all the air in the pleural space is reabsorbed and the lung fully expanded. Severe respiratory embarrassment caused by pneumothorax indicates that increased pressure exists in the pleural cavity. Such a tension pneumothorax is an emergency requiring immediate release of the pressure by a needle introduced through the chest wall. If the tension pneumothorax recurs, pleural drainage by catheter to a water-seal or the use of continuous suction may be necessary.

The respiratory movements are also studied for signs of the typical gasp-like breathing, tracheal tug and lack of intercostal muscle activity which are seen in patients still partially paralyzed by muscle-relaxing drugs. If such signs are found and the drug used has been a short-acting depolarizing agent such as succinylcholine, a period of assisted respiration is all that is usually necessary. If curare is the cause of impaired breathing, neostigmine with atropine or edrophonium (Tensilon) may be used to return the respiration to normal. A second dose of these antidotes may be necessary to prevent the signs of paralysis from returning as the action of the first dose wears off.

If respiratory movements are smooth, symmetrical and unobstructed, the rate and depth are ascertained to make sure that pulmonary exchange is adequate. Since ventilation of the alveoli is the essential purpose of external respiration, increased depth can compensate for a diminished rate, although an increased rate is not nearly so efficient in compensating for shallow respiration. Shallow respiration may do little more than ventilate the dead space and leave the alveolar air almost unchanged. Diminution of either rate or depth when respiration is smooth is a sign of considerable central depression. A slow rate is more typical of depression by narcotic agents, while almost all other central nervous system depressants predominantly reduce the depth. We now have reliable antagonists to narcotic depression of respiration in N-allylnormorphine and levallorphan but in the absence of narcotics these agents can themselves act as respiratory depressants.[4] At present the only reliable antagonists of the non-narcotic depressants are time, the stimulating effect of postoperative pain and the return to consciousness.

Not only can several of the causes of inadequate postoperative ventilation be present in the same patient but some of these causes can predispose the patient to others. Thus a partially paralyzed, depressed patient is more likely to have an obstructed airway or to develop atelectasis.

A further problem is presented by apnea that

continues into the post-operative period. Although complete control of respiration is rarely a necessity when balanced anesthesia is used, assistance of respiration is often advisable. When respiration is assisted, many patients cease making spontaneous efforts and passively allow themselves to be ventilated. The end of the operation finds these patients no less apneic than they would be from apnea deliberately brought about as a necessary part of the operative procedure.

In managing postoperative apnea one must first determine the cause. The amounts of the various agents used during the procedure, and the time of administration, are reviewed, as well as how the patient responded to each drug when it was administered. An observation of exceptional sensitivity to any of the drugs used may help incriminate it as the cause of apnea.

The first step toward making a diagnosis is the elimination or "washing out" of inhalation agent. This may so reduce the level of analgesia that painful stimuli stir the respiratory center into activity. Having done this, one must make certain that he is not faced with mere "breath holding." The presence of an endotracheal tube or an oro-pharyngeal airway may be the stimulus for breath holding. The "breath holder" resists efforts made to expand his lungs fully and also demonstrates generalized increased muscle tone. Such patients soon resume spontaneous respiration.

If the postoperative apnea is not the result of breath holding and if the patient has been subjected to controlled respiration during a long operation, habit apnea can be suspected. This suspicion is strengthened if the dosage of the drugs used was relatively small. Continued rhythmic controlled respiration tends to prolong the apnea. Oxygenation of the patient should be maintained by irregular compression of the rebreathing bag. If oxygen is used for this purpose, some accumulation of carbon dioxide is permissible but only to the point of allowing a slight increase in the pulse rate. When spontaneous respiration reestablishes itself, the cause of the apnea is confirmed if the patient progresses from small gasps at a slow rate to a full depth and rate in a matter of minutes.

There now remain for discussion those patients with apnea that is either the result of excessive sedation or paralysis by the muscle-relaxing drugs. When sedation is at fault, which is suggested if an excessive amount of sedative agents have been used during the operation, treatment can begin by counteracting the effect of the narcotics used. Both N-allynormorphine and levallorphan are effective in antagonizing the respiratory depression of narcotics. If barbiturates are the cause of the lack of spontaneous respiration, there are as yet no reliable proved antidotes although much work is reported in this field and several promising agents have been tried.[7] A central nervous system stimulant such as caffein sodium benzoate, although not specific, may be used with some success. Brief duration of action is the main disadvantage. By themselves the doses of the barbiturates used in the operating room seldom produce apnea of long duration, but the ensuing inadequate ventilation may require supportive treatment for many hours.

Total paralysis caused by the muscle relaxants such as curare and succinylcholine should be suspected as the cause of apnea if large doses of these drugs were necessary during the operation. This occurs most frequently during long operations in the upper abdomen. Artificial respiration is continued until some spontaneous efforts are observed, at which time antidotes may be administered. Edrophonium and neostigmine with atropine are useful in counteracting the paralysis of the curare group of relaxants but they may accentuate the paralysis of the depolarizing agents such as succinylcholine. Second and third doses of the antidotes at half hour intervals may be necessary to maintain their effectiveness. Although there are no antidotes for succinylcholine and related relaxants of the depolarizing group, the period of action of these drugs is briefer after the first spontaneous movements are observed. Some cases of apnea following intraperitoneal administration of neomycin have been reported, but they responded to neostigmine treatment.[6]

Anyone who uses muscle relaxants often enough will sooner or later encounter the complications of postoperative apnea or partial respiratory paralysis of many hours' duration, despite the use of antidotes. The patients in such cases may have a potassium deficiency and there are now reports of successful treatment of this condition with intravenous potassium.[5] The treatment is administered by slow infusion of a 0.3 per cent solution of potassium chloride in 5 per cent dextrose in water or in one-half normal saline solution, the rate of infusion being controlled by monitoring the electrocardiographic tracings for signs of potassium toxicity. Perhaps this treatment will prove to be consistently effective in those cases of apnea that do not respond to the usual care.

In all instances of prolonged apnea, proper ventilation is the main consideration. Hypoxia, hypercapnia and hypocapnia can each depress the respiratory center and thus prolong the apnea, although each by different mechanisms of action.[8] Controlled respiration that will provide adequate oxygen supply without seriously altering the carbon dioxide level is essential. If carbon dioxide analyzers were

part of every anesthetic machine, one could routinely monitor the carbon dioxide tension of the expired air. For practical purposes, if air instead of oxygen is used to ventilate an apneic patient's lungs and the minute volume is just great enough to prevent cyanosis, the carbon dioxide level will not veer too far from normal. This can be done with a Kreiselman resuscitator or with an anesthetic machine that has a source of compressed air.

There is usually more than one cause of postoperative respiratory inadequacy. Treatment consists first of establishing patent air passages and providing adequate ventilation. Although stimulants and antidotes may be available, they supplement rather than replace controlled or assisted ventilation of the lungs. A patient who can just maintain oxygen requirements while breathing a mixture with a high oxygen content cannot be left on his own. Such breathing is not sufficient to eliminate carbon dioxide and may also permit hypoxia if the patient should slump back into a more deeply sedated state. The danger point may be considered passed when the patient is able to satisfy his oxygen requirements for more than five minutes while breathing room air unassisted. A patient at that stage, although still showing some signs of depression or paralysis, can be sent safely to a recovery room to have oxygen administered by catheter, mask or tent.

Problems in ventilation in the immediate postoperative period occur very frequently. Prompt recognition, diagnosis and treatment are essential for the prevention of serious or permanent complications.

University of California Hospital, San Francisco 22.

REFERENCES

1. Brodie, Bernard B., Burns, J. J., Lief, Phillip A., and Papper, E. M.: Intravenous anesthetics—New concepts, Anesth. & Analg., 31:145, May-June 1952.

2. Burstein, C. L.: Fundamental Considerations in Anesthesia, The Macmillan Company, New York, 1955.

3. Foldes, F. F.: Muscle Relaxants in Anesthesiology, Charles C. Thomas, Publisher, Springfield, Ill., 1957.

4. Foldes, F. F., Zeedick, F. J., and Koukal, L. R.: The effect of narcotic analgetics and narcotic antagonists on respiration, Am. J. Med. Sc., 233:153, Feb. 1957.

5. Keating, V., and Tang, K.: Potassium deficiency and neostigmine resistant curarization, Anesth. & Analg., 36:32, Nov.-Dec. 1957.

6. Middleton, W. H., Morgan, D. D., and Moyers, J.: Neostigmine therapy for apnea occurring after administration of neomycin; report of a case, J.A.M.A., 165:2186, Dec. 28, 1957.

7. Plum, F., and Swanson, A. G.: Barbiturate poisoning treated by physiological methods, with observations on effects of beta, beta-methylethylglutarimide and electrical stimulation, J.A.M.A., 163:627, March 9, 1957.

8. Rayburn, D. J., Whitehead, R. W., and Draper, W. B.: The influence of respiratory acidosis on the plasma levels of thiopental and on the depth of anesthesia, Anesth. & Analg., 32:280, July-August 1953.